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A STUDY IN WOUND BALLISTICS

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The wound ballistics problem is divided into a series of subproblems. A specific definition of each subproblem is chosen by quantification of biophysical, physiological, and pathological variables that are relevant to the problem. The choice of the quantification leads to a number of hypotheses about functional relations among statistics of quantified variables. Experimental and observational data for testing the proposed hypotheses are discussed. Two aspects of synergism of multiple wounds are discussed and an approach for estimation of their effects is proposed. The first of these two aspects concerns biomechanical interaction of physical impairments inflicted by

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wounds. The second aspect is the interrelation between systemic response to injury and the wounds. Serious and lethal wounds are discussed in terms of morbidity and mortality. Mathematical models for mortality and morbidity are proposed.

PREFACE

The work described in this report was authorized under Project/Task 1T062110A027. This work was started in July 1971 and completed in January 1972.

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CONTENTS

I. INTRODUCTION.

Wound ballistics seeks to relate ballistic properties of a projectile to effects of a hit. The complexity of the phenomenon calls for a subdivision of the problem into simpler subproblems and for an investigation of special cases of these subproblems. A solution of the general problem can be obtained by extending the relations obtained for the special cases and synthesizing a solution from these partial results. The general problem can be divided into subproblems in many different ways. Each subdivision may allow a variety of approaches to a synthesis of a general solution. One such subdivision and a method of synthesis can be readily recognized in the approach used in the past.[1]

The past studies began with the selection of a projectile; i.e., its shape, mass, and striking speed. A series of projectile hits was examined by determining the rate of retardation in various mammalian tissues and by tracing each path on charts of the human anatomy. The resulting injury was estimated by comparing these paths with qualitative information on injury produced in experimental animals. The injury was interpreted in terms of percentage of incapacitation. Thus, we have a subdivision of the problem into a description of the projectile, a study of hits, an estimation of injury, and an assessment of incapacitation.

The past investigation was restricted to certain projectile shapes and velocities, and only horizontal trajectories through the body were analyzed. The results obtained for several combinations of these variables were synthesized into percentage of incapacitation from which equations for probability of incapacitation were obtained.[1]

In this report, we propose to retain this subdivision of the problem into ballistic properties of the projectile, the hit, the injury, and the consequences. We think that the wound ballistics problem will benefit from a subdivision into still smaller problems and from an explicit statement of selected restrictions and intended extensions. A detailed list of all the constituents of this subdivision, together with a precise definition of every element, shows quite readily the possible choices of subproblems that can be obtained by restrictions to special cases. At the same time, the list of constituent parts puts each subproblem into proper relation to other subproblems. We can quickly see which elements can be quantified and related analytically to each other and which relations should remain qualitative, expressed in a form of branching lists or tables. As we cannot establish empirical relations between every projectile and the corresponding consequence, our final relations must be analytic or at least they must be quantitative to permit a meaningful extension in terms of interpolation or extrapolation; however, intermediate relations may well be of a tabular or a similar nature.

This report does not present solutions to any subproblem of wound ballistics. Instead it is limited to an analysis of the problem. We believe that, whenever possible, analysis should precede the experimentation and data collection, both of which are indispensable for a solution. Analysis is simply an explicit enumeration of the assumptions, restrictions, and hypotheses that define subproblems in a sufficiently simple and specific manner in order to permit a feasible approach and a solution within a reasonable time. One should start collecting experimental data for testing the hypotheses and for constructing a solution only after specific hypotheses have been formulated, and these should approximate the complex reality by simplified relations. Some hypotheses proposed in this report can be tested with the data that are already available.

We believe that the analysis presented below puts in a proper perspective the problem of the synergistic effect of multiple wounding as well as the question of serious and lethal wounds.

II. PROBLEM ANALYSIS.

As stated in the introduction, we divide wound ballistics problems into a description of the projectile, the hit, the injury, and the consequences of the injury. It is not possible nor necessary to provide a complete description of these four parts of the phenomenon; rather we need only to specify those aspects which are essential.

In other words, we must define abstract concepts of a projectile, a hit, and others, which are specific in their relevant aspects and, at the same time, are general by explicit exclusion of irrelevant properties such as the color of a projectile. What is relevant and what is not depends on the purpose for which a solution of the problem is sought. We begin with the consequences that seem to be of immediate relevance to the military. Incapacitation is an important consequence relative to munitions effectiveness and casualty production. However, in the design of protective gear for personnel, a definition of serious and lethal wounds is a more desirable measure of effectiveness for describing reduction in casualties. In this context, it seems that mortality and morbidity may be appropriate criteria for classification of wounds into lethal and nonlethal and serious and nonserious. Consequently, we choose mortality, morbidity, and incapacitation as consequences of injury.

With this in mind we partition injury into the local tissue damage of the wound and the systemic response; i.e., the change of various physiological functions induced by the wound. The local damage depends primarily on the forces acting on the tissue, whereas systemic response is mostly dependent on the damage to the physiological system. Therefore, the description of a hit must include both the anatomical site and the force exerted on the tissue.

The force depends, among other things, on the total energy of a projectile and its shape. We specify the projectile by its mass, striking speed, shape, and type.

On the basis of the preceding, we obtain the following first level subdivision of the problem.

1. Projectile
   - Mass of projectile
   - Impact speed
   - Shape of projectile
   - Type of weapon

2. Hit
   - Anatomical site
   - Interaction force

3. Injury
   - Wound
   - Systemic response
   - Feedback between wound and response

4. Consequences
   - Mortality
   - Morbidity
   - Incapacitation

In this subdivision, only the mass and the speed of a projectile need no further specifications, whereas the remaining concepts still must be defined. It is useful to subdivide all these elements further; i.e., to enumerate their parts and then to define each part.

In order to define the shape of a projectile, we propose a first approximation based on the assumption that the projectile does not break up or deform upon impact. Therefore, the shape of a projectile with a regular geometry can be readily described in terms of a few numerical parameters; for example, as the radius of a sphere. An irregular projectile such as a fragment can be approximated by an ellipsoid or parallelepiped. The most convenient choice of parameters depends on postulating wounding mechanisms which include an assumption on the interaction between the projectile and the tissue.

A hit is described by an anatomical site, such as the head, thorax, abdomen, or limbs. In each anatomical site, we have at least two kinds of tissue: soft (skin, fat, muscle, organ parenchyma, fluid, air) and hard (bones, cartilage, ligaments). Therefore, a description of a hit also involves the force of interaction between the type of tissue in the anatomical site and the projectile. This force is a function of time or distance, as well as of projectile characteristics and physical properties of the tissues.

In order to relate the injury to the consequences, we must describe the wound by anatomical site and also we must specify the physiological systems involved, such as central nervous, cardiovascular, pulmonary, and others. The systemic response parameters and their changes are a part of description of the injury.

Mortality can be expressed either as time to death after injury or as rate of mortality within a specified period. In different applications of the wound ballistics model, we may be interested in either of these descriptors; hence, we include both in our definition of mortality.

We choose to measure morbidity in several ways. It can be measured in terms of general derangement of the injured, the urgency for medical attention, the recovery period, or permanent disability.

Examples of levels of incapacitation are probabilities of incapacitation related to various combat missions, as discussed in reference 2, for instance.

Specific definitions of all these components of our subproblems are presented below together with a discussion of their interrelation.

III. CONSEQUENCES.

As stated above, we are interested in the following consequences of injury: mortality, morbidity, and incapacitation. Mortality can be expressed as the expected mortality rate within a specified period of time or as the expected time to death. Abstractly, mortality is a pair that consists of a function of period of time (expected mortality rate) and a scalar (expected time to death).

Of course, more generally, we should consider mortality rate as a random function and time to death as a random variable instead of their expected values. However, this would complicate appreciably our problem that is already difficult, since it would require determination of the respective probability distributions. Instead, we simplify the problem even further and replace the function of time that expresses expected mortality rate by its values at preselected points, say, $t_1, t_2, \ldots, t_n$. These may be, for instance, as follows: $t_1 = 30$ sec, $t_2 = 5$ min, $t_3 = 30$ min, $t_4 = 12$ hours, $t_5 = 5$ days, or any other collection of interest. Thus, we define mortality as a six-dimensional vector whose components are five expected mortality rates within specified periods of time and the expected time to death.

Morbidity can be defined as the deviation of an injured person from normal. We call this deviation derangement. It can be measured in terms of the changes in physiological functions such as ventilation, circulation, metabolism, endocrine secretion, and neurological functions. Obviously, this deviation is a function of time and, as in the case of the rate of mortality, we replace this vector-valued function by its values at specified instances of time.

Another measure of morbidity is the time interval that an injured person can wait for medical treatment without an appreciable effect on his chance for recovery. We call this tolerance period.

The third measure of morbidity is the expected hospitalization period which we call recovery time.

We are also interested in a long-range effect of injury. Hence, we include permanent disability as a part of our definition of morbidity. The Committee on Rating of Mental and Physical Impairment divides the problem of determination of disability into the medical and administrative parts. The medical problem consists of determination of permanent impairment; i.e., a change in physiological and biomechanical functions due to damage to organs and

tissues. The administrative part is an interpretation of impairment in terms of disability. Besides the dependence on impairment, the resulting disability is influenced by the age of the person, by his working conditions, his adaptability, and by other factors.

The Committee published Guides to Evaluation of Permanent Impairment,[2-12] which enumerate impairments and the associated symptoms and pathology in the following areas of medical interest:

1. Ear, nose, throat, and related structures
2. Extremities and back
3. Visual system
4. Cardiovascular system
5. Central nervous system
6. Digestive system
7. Peripheral spinal nerves
8. Respiratory system
9. Endocrine system
10. Mental illness
11. Reproductive and urinary system

Impairments and related pathological conditions are further delineated in these guides. For instance, an impairment of extremities includes deficiencies in fingers, hands, arms, legs, etc., as separate impairments. The guides also provide a conversion of impairments into percentage of disability. Thus, these guides can be used to express this aspect of morbidity as percentage of permanent disability.

The third consequence of injury is incapacitation; i.e., reduction of ability to perform specified military tasks such as assault, defense, supply, and reserve. We feel that this consequence should be divided into medical and administrative (tactical) parts in a fashion similar to the permanent disability discussed above. The medical problem again is a determination of the impairment of physiological and biomechanical functions. We define this as a present impairment, in contradistinction to the permanent impairment discussed above.

We may begin by refining the functional groups that are being used in the studies of incapacitation and consider the level of consciousness and the ability to communicate as separate functional groups instead of expressing them in terms of functions of extremities as is currently done. We may further refine the groups that constitute a part of the presently employed assessment of incapacitation by adding separate groups for loss of dexterity of fingers and hands. An analysis of activities required in the performance of selected combat duties may also be very useful in choosing a proper refinement of the present groups.

Another approach to a definition for present impairment is to begin with the list of permanent impairments which can be constructed from information contained in the guides[2-12] and modify this list to obtain a meaningful collection of present impairments.

In any case, we would define present impairments either by constructing a list of the familiar 16 functional groups, or a refinement of these groups, or by tabulating a more comprehensive grouping comprised of the various cases analogous to permanent impairments enumerated in the guides.[2-12]

The list of present impairments must be supplemented by formulas and tables that convert the impairment into incapacitation in a fashion similar to that for converting a permanent impairment into permanent disability as described in the guides.[2-12]

At this point, it should be noted that there are two types of synergistic effects of impairments. One is due to direct interaction of impaired biomechanical functions and the other is due to the feedback between the wound and systemic response. The first type of effect is analyzed in the guides;[2-12] and, according to the rules for converting impairments to disability as formulated in these guides, the results of some impairments are added. There

are impairments that jointly produce less disability than one would obtain by simply adding the results of each impairment, and there are also impairments that jointly produce more disability than just the sum of individual contributions. All this must be considered in the evaluation of synergistic effects of multiple present impairments. Of course, the feedback effect mentioned above must be considered additionally.

IV. INJURY.

There are four aspects of injury: the wound, its local effect, the systemic response, and their interaction. By the wound we mean a disruption of structural integrity of the injured tissue and the histopathological conditions. Systemic response consists, among others, of a change in blood pressure, heart rate, endocrine secretion. We must also consider the local effect of the wound and the feedback between the wound and the systemic response.

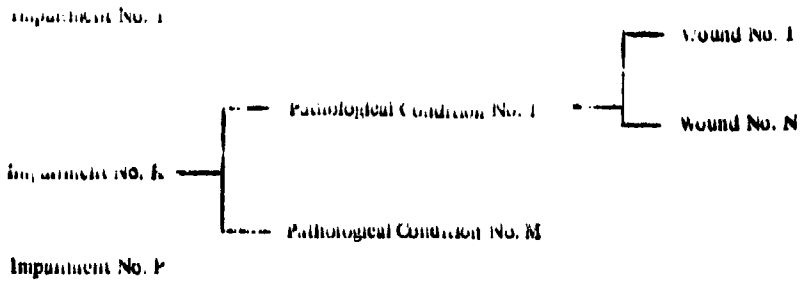
A specific description of the wound and of the systemic response depends on the consequences that we want to consider. Therefore, we discuss the injury in relation to each of the three aspects described above.

Mortality is a result of a change of physiological functions; therefore, we need only to consider systemic response measured in terms of disruption of homeostasis. Immediate change in homeostasis is the change of hemodynamics and ventilation. This, in turn, affects the response of the endocrine and nervous systems. The result of all of these changes is an alteration of blood gases and metabolism which affect hematological parameters and integrity of cells and, consequently, the level of serum enzymes. Therefore, we can describe the systemic response in terms of parameters that characterize the following: (1) hemodynamics, (2) ventilation, (3) endocrine and sympathomimetic secretion, (4) blood gases, (5) serum colloids, (6) metabolites, (7) enzymes, (8) hematology, and (9) coagulation.

Numerous efforts have been made to determine mortality rate (probability of death) in terms of the variables listed above. For instance, the probability of death is obtained as a function of a single metabolite, namely, lactate, by Weil and Afifi.[13] It was shown that, for a group of 142 patients suffering shock of varying etiologies, the prediction of survival and nonsurvival based on this parameter was 88% accurate.

Afifi *et al.*[14] chose a set of successive determinations of arterial systolic blood pressure (hemodynamic parameter) and pH (blood gas) as predictors of the outcome of 52 patients that were in shock because of overdoses of barbiturate, glutethimide, or meprobamate. A discriminant function based on these variables computes mortality with 86% accuracy. Thus, various linear and nonlinear discriminators are available that can be used to determine mortality rate in terms of systemic parameters. Expected time to death can also be estimated on the basis of the same variables. We discuss specific models for these variables in section VII.

Morbidity aspects are of two categories. Derangement, tolerance period, and recovery time belong to the first category and can be expressed in terms of systemic variables in a fashion similar to mortality. The second category includes permanent impairments. These depend on a wound and on the systemic response. A description of wounds that can be readily related to permanent impairment might be obtained as follows. First we list all the permanent impairments described in the guides.[2-12] For each impairment, we list pathological conditions explicitly mentioned or implied. Next, for each pathological condition, we describe a class of wounds that lead to the conditions. Thus, we consider the following scheme:



It should be noted that the relation of a wound to the permanent pathological condition may be stochastic; i.e., we may consider probabilities of several pathological conditions together with specification of a wound. Similarly, permanent pathological conditions may be related to permanent impairments in terms of probabilities.

The sublists of pathological conditions extracted from the guides[2-12] should be examined for adequacy and supplemented, if necessary, by a procedure based on the Delphi method; i.e., proper questionnaires should be designed and the answers should be collected and edited according to the Delphi technique. The same procedure should provide sublists of wounds for each permanent pathological condition. This would yield a relation between permanent impairments and the wounds.

The next step would be to collect accident and battlefield data of wounds that are included in the sublists and check the validity of the relation. Obviously, we cannot hope to collect enough data for checking every branch of the list of impairments, pathological conditions, and wounds. Therefore, most of the relations will remain subjective assessments. However, they will be objective in that they will represent a pooled opinion of several assessors and will remain the same for each application until revised and improved.

Present impairment in the incapacitation should be handled in a similar manner. Again one should begin with a list of impairments of biophysical functions, including communication and consciousness. The sublists of pathological conditions for each impairment and the sublists of corresponding wounds should be constructed. A relation of a wound to systemic response should be taken into account by specifying pathological conditions for various periods of time. In other words, for a specified impairment such as loss of dexterity of digits, we may choose several pathological conditions of varying severity according to the period of time between wounding and the manifestation of the impairment. It may be expedient to consider aggravation of the impairment by derangement obtained in the assessment of morbidity. Such a relation of physical impairment to the level of derangement may be a proper vehicle for introducing the synergistic effect of multiple wounding.

The validity of the relation between the present impairments and the corresponding wounds should be tested by available accident and battlefield data.

In summary, the injury consists of the values of systemic variables determined at a specified time after wounding, the description of a wound in terms of damage to structural integrity, and pathology. The type of tissue and the organ or physiological system involved must also be specified in this description. Wound description may remain qualitative. However, it may be expedient to quantify the wounds.

We may quantify a wound by a triplet of numbers as follows. Let w_1 be the total volume of tissue that must be removed before suture of the wound. We call the cavity that results from debridement (assuming no contraction or expansion of the remaining tissue) the ultimate cavity. For brevity, we also call its volume, w_1, ultimate cavity. Let w_2 be the maximum cross-sectional area of the ultimate cavity. We call w_2 ultimate cut. These two quantities correspond to two different wounding mechanisms that are frequently referred to as energy deposition and cutting. Histopathological conditions are quantified as follows. After debridement, the wound is flushed with saline and the washed-out tissue cells are spun off for a dye-exclusion test of viability. The percentage of the dye-exclusion cells is determined. Let this percentage be w_3. Thus a complete definition of the wound for each type of tissue requires a triplet of numbers: w_1, w_2, and w_3.

One can expect a considerable variability in the estimates of these numbers, especially because of variability in judging how much tissue should be debrided. In animal experiments, the procedure for debridement can be prescribed in detail in order to reduce this variability, which also affects the histopathological value w_3. One possible approach may be to use the change in color of liquid crystals sprayed on the injured tissue in order to determine the amount of tissue to be debrided. Experiments at the Biophysics Division using this technique suggest that it may be profitable to search for a refinement of this method. A refi[redacted] may [redacted] effecting relatively uniform debridement.

Experimental data may be employed to obtain an analytic approximation of ultimate cavity and ultimate cut in terms of the force exerted on the tissue. Let us assume that the postulated wounding mechanism leads to two different types of interaction forces, say, F_1 and F_2. In the next section, we discuss a hypothesis that leads to two such forces. We may try to represent the ultimate cavity, the ultimate cut, and histopathology in terms of functionals of these forces. Let us assume that the area $A(t)$ of the cross section of the ultimate cavity normal to the path of the projectile is proportional to F_1, and the maximum diameter of this cross section, $D_{max}(t)$, is proportional to F_2; i.e., $A(t) = aF_1$ and $D_{max}(t) = bF_2$. Then the formulas

$$w_1 = \int A(t)v(t)dt \tag{1}$$

and

$$w_2 = \int D_{max}(t)v(t)dt, \tag{2}$$

with integration over the duration of penetration, approximate the cavity w_1 and the ultimate cut w_2. The quantity w_3 may be attributed to hydrodynamic force only and hence we may consider the following approximation:

$$w_3 = c\int F_1(t)dt \tag{3}$$

where C is a constant.

This choice of quantification of a wound requires that the questionnaires discussed above be complemented with autopsy pictures and the histological findings corresponding to the various levels of damage. This information should be associated with the corresponding values of w_1, w_2, and w_3. The questionnaire would also require the values, or ranges of values, of w_1, w_2, and w_3 to produce the pathological conditions contained in the lists of permanent and present impairments. Of course, all experimental data, including autopsy and histological records, should first be examined to test if the proposed quantification of wounds is adequate.

A simple and yet sufficiently accurate description of local injury seems to be the most complicated part of the wound ballistics. An extensive analysis of existing experimental data, including tissue simulants, should be conducted and a few simple hypotheses on interaction mechanisms between a projectile and the tissue should be tested. Some new experiments, especially with tissue simulants, may be required for determination of an acceptable interaction model.

V. HIT.

Description of a hit depends on the choice of quantification of the injury. We introduced a concept of sublists of pathological conditions that lead to a present or permanent impairment. These conditions are associated with certain anatomical parts of the body. Hence, for a specified part, protective environment, and weapon or weapon system, we can determine the conditional probability of a hit of that anatomical part, given a hit of the body. Conditional probabilities for several anatomical parts may be greater than zero for certain single hits.

The wound is described in terms of disruption of structural integrity of the tissue and in terms of induced histopathological conditions. Therefore, a hit should also include a conditional probability of exerting that force which produces the damage. Thus, we need to determine an interaction mechanism between projectile and tissue and derive from this relation the frequencies of various magnitudes of the interaction force. We choose the following hypothesis: the force consists of two components, one being of hydrodynamic drag type and the other of elastic-plastic reaction type. Thus we propose to test the following simple equation of motion of a projectile within the tissue:

$$m\frac{dv}{dt} = -c_1v^2 - c_2v \tag{4}$$

where m is the mass of the projectile, v is its speed, and c_1 and c_2 are proportionality coefficients that depend on properties of tissue and projectile, such as shape and orientation. If, for each type of tissue, we replace c_1 and c_2 in equation 4 with their expected values taken over all the possible orientations of a projectile, we can integrate equation 4 to obtain

$$v = v_0 e^{-\frac{c_1 x}{m}} - \frac{c_2}{c_1}\left(1 - e^{-\frac{c_1 x}{m}}\right) \tag{5}$$

where x is the depth of penetration and

$$\frac{c_2}{v_0} e^{\frac{c_1 x}{m}} = \frac{c_2 + c_1 v_0}{v_0} - e^{-\frac{c_2 t}{m}} \tag{6}$$

where v_0 is the striking velocity. Now we can obtain the two forces (their expected values) of the interaction between the projectile and the tissue, namely, $F_1 = c_1v^2$ and $F_2 = c_2v$, as functions of time.

We can obtain a description of a hit either by examining available battlefield data or by comparing the geometry of the involved anatomical part with the geometry of the missile path and, if applicable, with protective barriers.

The data on projectile retardation by mammalian tissue and by tissue simulants can be used to test the interaction model and to determine its parameters such as c_1 and c_2 in equations 5 and 6.

The model and the geometry of the tissue involved can be used for determining probability of the interaction force or of some functional of this force such as, say, total work (energy deposited) or total momentum. Of course, these parameters of hit depend on the projectile descriptors.

VI. PROJECTILE.

A complete description of the projectile should include joint frequency distribution of its mass, speed, and shape parameters. However, for the sake of simplicity, we propose to consider the mean values (or rather their estimates) of projectile descriptors. This remark completes our definition of the mass and the striking speed of a specified projectile at a specified range.

For definiteness, we assume that the shape of a projectile is described by four parameters. The first parameter is the maximum presented area; the second, the maximum diameter of this area; the third, the minimum presented area; and the fourth, the maximum diameter of the minimal area. The sphere of radius r has, accordingly, the following shape parameters: πr^2, 2r, πr^2, and 2r. The cube of edge a is characterized by the following parameters: $a^2\sqrt{3}$, $a\sqrt{3}$, a^2, and $a\sqrt{2}$. The shape parameters of a parallelepiped of dimensions a × b × c with $a \geq b$ and $b \geq c$ are: $\sqrt{a^2b^2 + b^2c^2 + a^2c^2}$, $3abc/\sqrt{a^2b^2 + b^2c^2 + a^2c^2}$, bc, and $\sqrt{b^2 + c^2}$. The shape parameters of any projectile of regular geometric form can be expressed in terms of its geometry. However, it is simpler to derive these parameters empirically. This can be done by obtaining the two orientations of the projectile that produce the smallest and largest shadows, with the projectile placed in the path of parallel rays. Such orientations can be readily determined with the aid of a photometer. Measurement of areas and maximum diameters of these shadows produces the desired parameters. The shape parameters of bullets and flechettes can be readily approximated in terms of their geometric parameters. For instance, if the stem of a flechette has radius r and length ℓ and two rectangular fins of dimension a × b × c, then the shape parameters of the flechette are, approximately, $2(r\ell + ab)$, ℓ, $\pi r^2 + 2bc$, and $2(b + r)$.

The choice of these shape parameters is dictated by our hypothesis concerning the relation between projectile descriptors and the hit characteristics. Our hypothesis assumes that, for a fixed type of tissue and a fixed type of projectile, i.e., projectiles with the same stability properties, the coefficients c_1 and c_2 in equations 5 and 6 can be approximated as follows:

$$c_1 = \alpha_1\rho_1 + \alpha_2\rho_3 \tag{7}$$

and

$$c_2 = \beta_1\rho_2 + \beta_2\rho_4 \tag{8}$$

where ρ_1, ρ_2, ρ_3, and ρ_4 are the missile shape parameters defined above. The coefficients α_1, α_2, β_1, and β_2 depend on mechanical properties of tissue and on the stability of the projectile. If c_1 and c_2 are determined for several shapes by the method described in the preceding section, we can use equations 7 and 8 to compute α_1, α_2, β_1, and β_2 by the least squares method or some other estimating technique. If the hypothesis is changed in any way, then it may be expedient to choose different shape parameters. The ones chosen were selected because we propose to test our hypothesis.

We also consider the type of weapon as a part of the description of the projectile, since conditional probabilities of hitting various anatomical sites of the body may depend on the type of weapon.

VII. MORTALITY.

In the preceding sections, we attempted to describe the consequences, injuries, hits, and projectiles. Now we turn to relations between these elements. Let C be the set of consequences, W be the set of injuries, H be the set of hits, and P be the set of projectiles. The elements of these sets have been defined in the preceding sections with the following relations (mappings) in mind:

$$\begin{aligned} &h: P \to H \\ &w: H \to W \\ &c: W \to C \end{aligned} \tag{9}$$

The combination of these mappings produces a mapping of the set P into the set C; i.e., relates the descriptors of projectiles to the consequences. This is the ultimate goal of the wound ballistics which can be achieved if we succeed in deriving the three mappings indicated in equation 9.

In view of the complexity of these sets, especially of the set of injuries, W, it is advisable to examine a possibility of deriving mappings of simpler sets. For instance, instead of considering all of the complexities of the set W, we may study only the injuries to certain parts of the body, say, extremities. Suppose we succeed in obtaining a mapping of the set P (projectile descriptors) into a subset of consequences that result from injuries to extremities. This mapping can be used in two ways. First, we can use it to evaluate the relative effect of projectiles if we assume that whenever one projectile is, say, twice as effective on extremities as another, then it is also twice as effective in general. Secondly, the general mapping of P into C can be readily obtained from partial mappings of various subsets.

There is also another way of choosing partial problems that are much simpler than the general problem. These partial problems can be obtained by considering various projections of the sets P, H, W, and C. We recall that the elements of these sets are vectors (n-tuples of numbers). We can ignore some of the components of these vectors and consider the mappings of the sets of lower dimensions. We begin now with such a choice. Let V be the set of systemic responses to wounds and D be the set of mortality rates at a specified time t_0 after wounding. We seek the mapping of V into D.

The patient data recorded in the Center for Study of Shock and Trauma at the University of Maryland during the last several years establish our departure point in a search for such a mapping. These data contain various systemic parameters measured in shock and trauma patients. The patients are classified into survivals and nonsurvivals. The survivals are those who left the intensive care unit well enough to be transferred to a general hospital or to a rehabilitation center for physical therapy or to their homes. All nonsurvivals died in the intensive care unit.

Some of these patients came to the Shock-Trauma Center after unsuccessful treatment at other hospitals. For this and other reasons, the time of injury is not known in many cases. However, we postulate a model in which this is not important, because we assume that a sufficiently detailed record of the present state of every cell and every organelle in a cell does describe the condition of the patient so completely that his medical history cannot add anything to our knowledge of his condition. Of course, such a record is not available. However, we may test a hypothesis that a sufficient number of systemic variables can be selected so that the past medical history, including the time of onset of trauma and shock, is superfluous. If this hypothesis is correct, then the measurement of systemic variables at any instant should be sufficient for predicting whether the patient will die or live. On the basis of this hypothesis, we initially do not differentiate between causes of trauma and shock. This is consistent with our assumption that systemic response variables can be used to predict the mortality rate without reference to the wound that produced this response.

The patient data include 55 systemic variables. Our first step is to select a subset of these variables that is as small as possible and yet adequate for defining our mapping with sufficient accuracy. Therefore, we choose the so-called terminal point for each patient; i.e., the last determinations of the variables either before the death of the patient or before his discharge from the intensive care ward. The terminal sample should show the difference between near normal and poor prognosis conditions more clearly than any other choice of patient data.

Let x stand for an n-tuple (n-dimensional column vector) of systemic variables. We assume that the components of x are jointly normally distributed with the means $\bar{x}$ and $\bar{y}$ for survivals and nonsurvivals, respectively. Let M be the covariance matrix of the components of x. Then the likelihood ratio of x is

$$L(x) = \exp\{\tfrac{1}{2}[(x - \bar{y})^T M^{-1} (x - \bar{y}) - (x - \bar{x})^T M^{-1} (x - \bar{x})]$$

where superscript T denotes a transpose and M^{-1} is the inverse of M. Consequently, the probability that a patient will die with systemic variables x is

$$r^1(x) = \frac{1}{1 + L(x)}. \tag{10}$$

We selected 32 systemic variables that, by the Student's *t*-test, had the most significant differences between mean values for survivals and nonsurvivals. The data of 581 patients were used to estimate the mean values $\bar{x}$ and $\bar{y}$ and the covariance matrix M. Using these estimates, the probability $r^1(x)$ for each patient was computed by equation 10. If we assume that $r^1(x) < \frac{1}{2}$ indicates that the patient will survive and that $r^1(x) \geq \frac{1}{2}$ shows that he will die, then we find that this rule misclassifies 10% of the patients. Of course, this does not indicate that the estimate of probability is only 90% accurate. Indeed, a patient with the probability of 0.51 by this rule, for example, is classified as dead, yet he has 0.49 chance to survive. The accuracy of this probability must be tested by comparing the frequency of nonsurviving with the corresponding probability; that is, by comparing the expected number of nonsurvivals with the observed number of deceased patients. The sum of the probabilities computed for every patient yields the expected number of nonsurvivals. In our case, this sum is 238; i.e., the computed mortality rate is 0.4096 whereas the actual mortality rate is 0.4010.

This is only a preliminary result. The following steps must be taken to arrive at the formula for mortality rate. First, the normality of the distribution of each variable and the hypothesis on equality of covariances for survivals and nonsurvivals must be tested. Secondly, the selection of variables to be used in the model must be made according to their joint discriminating power instead of individual discriminating power as indicated by the statistical significance of the difference of the mean values or by some other criterion. Thirdly, other than terminal data should be used and the formulas for mortality rate during several periods of time should be derived. Finally, the dependence of the model on the cause of injury should be examined.

Successful completion of these steps will yield a mathematical model for mortality rate; i.e., an analytic formula that expresses the mapping of systemic vectors, x, into mortality rates, $r^1(x)$, for selected time intervals. Together with this we will obtain a collection of systemic variables that are adequate for relating a patient's condition to the corresponding mortality rate. Let X denote the set of vectors, x, of systemic variables, and let R be the set of k-tuples of mortality rates, r(x), in the specified time periods. Thus, we have

$$r: \mathbf{X} \rightarrow \mathbf{R} \tag{11}$$

The next step is to collect a set of human accident and battlefield data that include the needed information about the projectile, the hit, and the systemic variables, x, at a certain time after wounding. Suppose that we collect such data for several types of projectiles. The same type of injury can be simulated through animal experiments and the systemic response variables in the animal can be measured. For each $\rho \epsilon \mathbf{P}_1$, we get the animal systemic variables. We denote the collection of these variables by the vector g. Thus, for each $\rho \epsilon \mathbf{P}_1$, we obtain a pair (g, x). Hence, we can derive an empirical mapping, x = x(g), that maps a subset, G, of a set of all possible animal systemic variables, G, into a subset, X, of systemic variables in humans. By assuming that the derived formula, x = x(g), holds for every point, $g \epsilon G$, we obtain an extension ϕ of this mapping

$$\pi: \mathbf{G} \rightarrow \mathbf{X} \tag{12}$$

More generally, we may use suitable interpolation and extrapolation procedures to construct a mapping[12] from the pairs (g,x).

Now we can conduct animal experiments with projectiles and hits not included in the accident data and we can generate a number of triplets (p,h,g), where p is a set of projectile descriptors, h is a set of hit characteristics, and g is the set of animal systemic response variables. We can use these sets to obtain an empirical mapping

$$g = g(p,h) \tag{13}$$

The combination of this mapping with equations 11 and 12 yields r = f(p,h); i.e., the collection of mortality rates for a projectile, p, and the hit, h.

Let $h_1, h_2, \ldots, h_m$ represent a collection of hits that are of interest and let $q_1, q_2, \ldots, q_m$ be the probabilities of these hits. Then the mortality rate model for projectile p is given by

$$\rho(p) = \sum_{i=1}^{m} q_i f(p,h_i) \tag{14}$$

In order to complete the mortality model, we need to obtain a relation for expected time to death. We propose to derive this by modifying the Gompertz mortality law of actuarial mathematics. This law states that mortality rate m(t) (probability of death) at the age t is given by

$$m(t) = a \exp [b \exp (ct)], \tag{15}$$

where a, b, and c are model parameters. We assume that deterioration of the patient's condition is of the exponential type, as aging is; but the rate of deterioration is much faster than the rate of aging. Therefore, the same mortality law follows, except the time scale is different. Thus, we can obtain an empirical mortality law from equation 15 by changing the time scale. The new scale is a function of injury; i.e., a function of systemic variables. We denote the scale factor by k(x) with systemic variables, x, chosen at some fixed time. The resulting mortality law is:

$$\mu(t) = a \exp \left\{ b \exp[ck(x)t] \right\}. \tag{16}$$

Since the rate of deterioration increases with the severity of injury, we may obtain an adequate representation of k(x) in terms of r(x) which reflects the severity of injury. We may even limit ourselves to just one component of the vector r(x), say, $r_1(x)$. Since the scale factor increases at an accelerated rate as $r_1(x)$ goes to 1, it seems reasonable to expect that k(x) has a vertical asymptote at $r_1(x) = 1$ or at least an increasing slope as $r_1(x)$ approaches 1. Therefore, simple candidates for k(x) are either a reciprocal of a homogeneous polynomial in $1 - r_1(x)$ (or more generally in $(1 - cr_1(x))$ for some $c \leq 1$) or an exponential of this reciprocal; i.e., we write

$$Q(x) = a_0[1 - r_1(x)]^n + a_1[1 - r_1(x)]^{n-1} + \ldots + a_{n-1}[1 - r_1(x)] \tag{17}$$

with $a_{n-1} > 0$. We may choose either

$$k(x) = \frac{1}{Q(x)} \tag{18}$$

or

$$k(x) = ae^{\frac{1}{Q(x)}} \tag{19}$$

The expected time to death, T, is given by

$$T = \int_0^\infty t\mu(t)dt, \tag{20}$$

where $\mu(t)$ is defined by equation 16 with k(x) replaced by equations 18 or 19 or some other suitable function such as $k(x) = a_0[1 - r(x)]^{-\alpha}$, with $\alpha > 0$, but not necessarily an integer. Integration of equation 20 yields

$$T = T_0 k^2(x) \tag{21}$$

where T_0 is the expected lifetime with no injury. Substitution of actually observed data for $r_1(x)$ and survival time, T, yields an overdetermined system with coefficients of Q(x) as unknowns. The least squares solution of this system yields the values of the coefficients.

VIII. MORBIDITY.

A mathematical model for derangement and recovery period can be obtained in the same fashion as the mortality model described in the preceding section. In fact, derangement may be defined as probability of death which is a measure of deviation between the patient's present condition and his normal state.

A model for the tolerance period could also be obtained in a similar fashion if we could get data within this period. Obviously, the tolerance period cannot be measured directly. It can be obtained as an assessment by experienced surgeons. However, another way to estimate the tolerance period is as follows:

Suppose we have at least two determinations of systemic response, x, at the successive instances of time, say, t_1 and t_2, and that no effective therapy was instituted prior to the time t_2. With these and the mortality model, we can compute the mortality rates r_1 and r_2 at the times t_1 and t_2. By definition, the chance for recovery does not change appreciably during the tolerance period. Thus, the definition of the tolerance period must include a bound on this change. Suppose we allow the probability of death to increase during the tolerance period by the factor $1 + \epsilon$ for some small positive, ϵ. We use the values of r_1 and r_2 to extrapolate for the value r_0 at $t = 0$ and then for t_p so that $r(t_p) = (1 + \epsilon)r_0$. Then t_p is the tolerance period.

If the data as just described are not available in sufficient amounts for determining an empirical relation between systemic variables and the tolerance period, we may try to determine the effectiveness of the therapy instituted prior to t_2 and, consequently, to reduce the value of r_2 to discount this effect. The effectiveness of the therapy can be estimated by comparing the mortality rates of critically injured people brought to the hospital shortly after injury with the mortality rate in a similar patient group who arrive at the hospital with some delay.

Once the tolerance period for a number of patients has been estimated we can obtain an empirical formula that expresses this period as a function of systemic variables in a manner similar to that described in the preceding section.

The problem of permanent impairment and permanent disability is quite different since here the outcome depends on wound as well as on systemic variables. A model for permanent disability may require a series of formulas, at least one for each subsystem that constitutes a topic of any one of the guides.[2-12]

This problem is similar to the problem of incapacitation, which seems to be of a greater practical importance than the problem of permanent disability. Therefore, incapacitation should be addressed first and then the approach to the permanent disability should be patterned according to the method developed in the study of incapacitation.

IX. INCAPACITATION.

Evaluation of antipersonnel weapons, perhaps, depends on assessment of incapacitation much more than on any other consequences of the injury. Therefore, this part of the wound ballistics problem may be assigned the first priority. In view of the complexity of this part, a stepwise solution should be sought.

The present incapacitation models of Kokinakis and Sperrazza[1] express probability of incapacitation in terms of the mass and the velocity of a projectile. No estimates of the confidence intervals of computed probabilities are given. However, there are unpublished investigations of the confidence intervals. These results were obtained by at least two different approaches. Both are based on the assumption that the errors of log [-log (1-p)], where p is the probability of incapacitation, are normally distributed. This, perhaps, is a reasonable assumption. However, there are certain other assumptions in this study. In view of the scarcity of the data, it is not possible to obtain a reliable test of these assumptions. Therefore, it is desirable to obtain confidence intervals based on other assumptions and to examine the sensitivity of the model to these assumptions. The sensitivity of the model to the values of parameters of the assumed distributions should also be determined. The results of such a study should be published together with the confidence intervals of the past investigations and should be made available to the users of the incapacitation models.

The next step should be to assess the distribution of incapacitation levels associated with each functional group and to investigate the effect of this on the final model. If a definite incapacitation level is being assigned for each functional group as it was done in arriving at the models,[1] then the assumptions under which such an assignment holds should be examined and their implications should be investigated.

Similarly, instead of assigning a functional group to each wound class of the present approach, one should examine the probability distribution of functional groups as a function of the wound class. The effect of combining this distribution with the distribution of incapacitation levels should be investigated.

Also each missile trajectory traced on an anatomic chart should not be assigned a definite wound class. Instead, some probability distributions of wound classes should be associated with the trace of a projectile on an anatomical chart. A combined effect of all these probability distributions should be thoroughly investigated.

The probability distributions mentioned above can be obtained by applying the Delphi method to an appropriately devised questionnaire accompanied by the available experimental data. A much faster and easier approach would be to assume suitable ranges of the variables mentioned above and simple probability distributions over these ranges. For instance, instead of assigning the functional group X to a particular wound, we may say that the wound causes functional groups IX, X, and XI, all with equal probabilities of one-third or with some other probabilities.

Further, probabilities of particular traces should be reevaluated either on the basis of available field data or by considering more realistic relations between the position of the body and the path of a projectile, rather than an upright frontal exposure to horizontal paths of projectiles.

All that is said so far in this section concerns itself with a reevaluation of the existing experimental data and of the past analysis of these data. Additional studies should be conducted in a stepwise construction of new models. First of all, one may examine wounding mechanisms. To begin with a physiological effect, we may examine autopsy and histological records of the past experiments and attempt to determine if the total amount of tissue that should be debrided can be estimated from these records. The data of the past experiments also include retardation coefficients by various tissues. Therefore, it is possible to devise tissue simulants that produce the same retardation and to collect new experimental data with these simulants in order to obtain the forces postulated in a wounding mechanism as, for instance, F_1 and F_2 of section V. The tissue simulants could be used to obtain empirical relations between projectile parameters and the forces F_1 and F_2.

Determination of F_1 and F_2 or some other forces according to the assumed wounding mechanism for wounds of the past experiments will yield a tabular relation between these forces and the ultimate cavities and ultimate cuts. We can use this tabular relation to determine an empirical model that expresses the wound in terms of F_1 and F_2. If, as suggested above, F_1 and F_2 are expressed in terms of projectile parameters, we can obtain a mapping of these parameters onto a wound in a soft tissue.

In order to obtain at least partial results, which would constitute a first approximation to the desired solution, we may assess incapacitation induced by the wounds with specified ultimate cavity and ultimate cut only for soft tissues of gross anatomical components such as the head (brain), thorax, abdomen, and extremities. Instead of considering the effect of various projectiles, we examine the effect of various wounds and construct an empirical model of incapacitation that relates the ultimate cavity, the ultimate cut, and histopathology to incapacitation. The combination of this model with the previously described relation of projectile parameters to wound parameters yields a first approximation of the incapacitation model.

Consideration of skeletal structure as well as soft tissue, a finer subdivision of head, thorax, etc. into their anatomical components, and application of analysis described in this report would yield a better approximation to a desired model.

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